would appear to be a mechanical surgical problem involving control of the left coronary vein leading to the esophagus. It demands surgical consideration.

Ascites and anasarca often constitute the most difficult problems in polyserositis. In the majority of instances, immediate results can be obtained through the use of merbaphen and ammonium salts. The results of treatments with merbaphen in a series of six cases were reviewed by Snell (Fig. 18). In the subsequent control, under conditions at home, ammonium chlorid or ammonium nitrate may prove of value.

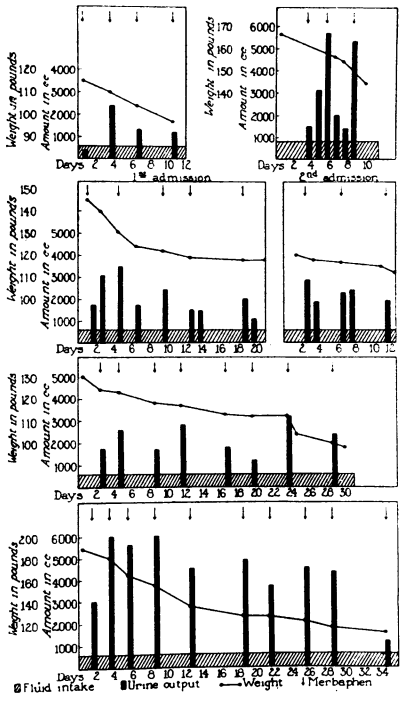

Fig. 18.—Results of treatment on four admissions of a patient with cirrhosis of the liver.

With the advent of the new diuretics a better selection than formerly is offered and better results are being obtained than ever before. There is still room for much progress in the treatment of edema. This progress may come through channels other than the administration of drugs such as are now in use. Some of the possibilities which have been suggested are denervation of the kidney by section of the lesser splanchnic nerves and stripping of the renal vessels, and the development of an active diuretic substance from the hypophysis or from the mammillary bodies. In this connection it may be noted that Bourquin has recently found it possible to get an extract from the mammillary bodies of dogs suffering from experimental diabetes insipidus which markedly increased the urinary output when injected into normal dogs.

COMMENT

I have discussed the most important diuretics and the various pathologic states in which they are indicated. But that is only the beginning. In practice, as we all know, each patient must be considered individually and each case of edema represents a distinct problem. I close, as I began, with a statement of Sydenham's with which I am sure all are agreed:

"The chief weakness of medicine is not our ignorance as to the ways and means by which certain indications may be satisfied, but our ignorance of the particular indications that thus want satisfying. How I can make a patient vomit, and how I can purge or sweat him, are matters which a druggist's shopboy can tell me off-hand. He can tell me, too, how to cool a man when he is heated. When, however, I must use one sort of medicine in preference to another, requires an informant of a different kind, a man who has no little practice in the arena of his profession."

The Mayo Clinic, Rochester, Minn.

CORONARY ARTERY DISEASE*

WITH REPORT OF CASES

By ROBERT WILLIAM LANGLEY, M. D.
Los Angeles

DISCUSSION *by Harry Spiro, M. D., San Francisco; Franklin R. Nuzum, M. D., Santa Barbara; Eugene S. Kilgore, M. D., San Francisco.*

INCREASING interest in the study of coronary artery disease during the last twenty years has led to more accurate diagnosis and to the early recognition of cases. The electrocardiograph has been particularly helpful in ruling out "acute indigestion," gall-bladder disease, and other vague abdominal pains. One is now able to cast a better prognosis and possibly prolong a patient's life by more accurate observation.

The cases given below represent a definite group of individuals who have survived attacks of acute occlusion of smaller branches of the coronary arterial system.

REPORT OF CASES

CASE 1.—Male, age sixty-six. On April 2, 1927, this patient was taken ill suddenly with a severe knife-like pain in the chest which was entirely substernal. The man felt as if he were going to die. He began to perspire and thinks he lost consciousness. He was a business man who had led a sedentary life for years and whose habits were moderate. On questioning him it was found that he had never had any serious illnesses that he could remember, and his only complaint had been a slight increased shortness of breath on unusual exertion during the last year.

The patient was examined in his office within one-half hour of the onset, and the pain relieved completely after a hypodermic injection of one-quarter grain of morphin sulphate. There was considerable pallor and cyanosis of the lips and ears, and perspiration was profuse. The pulse was 40 and the blood pressure, 146/90. No murmurs were heard and the heart was not enlarged to palpation. The patient was taken to the hospital immediately and an electrocardiogram made. The tracing revealed inversion of the T wave in Leads II and III, incomplete A-V block with migration of the pace-maker (Figure 1a). During this time the patient was in an orthopneic position constantly. Three grains of caffein sodium-benzoate was given every three hours during the day and, when necessary, at night. Morphin sulphate grains one-sixth was given twice. No digitalis was used. At the end of this period another tracing was made which indicated that the pace-maker had become stabilized. The T wave inversion was still present, showing a greater suggestion of the coronary arching as described by Pardee (Fig 1b). Several letters from the patient indicate that he has had slight precordial constriction on a few occasions during the last ten months, but has been otherwise perfectly well.

CASE 2.—Male, age sixty-one. In December 1926, the patient was referred by his son, a doctor, for an electrocardiogram. The tracing was without significant features in any lead and a report of a normal

* Read before the Innominate Society of Los Angeles, February 8, 1928.

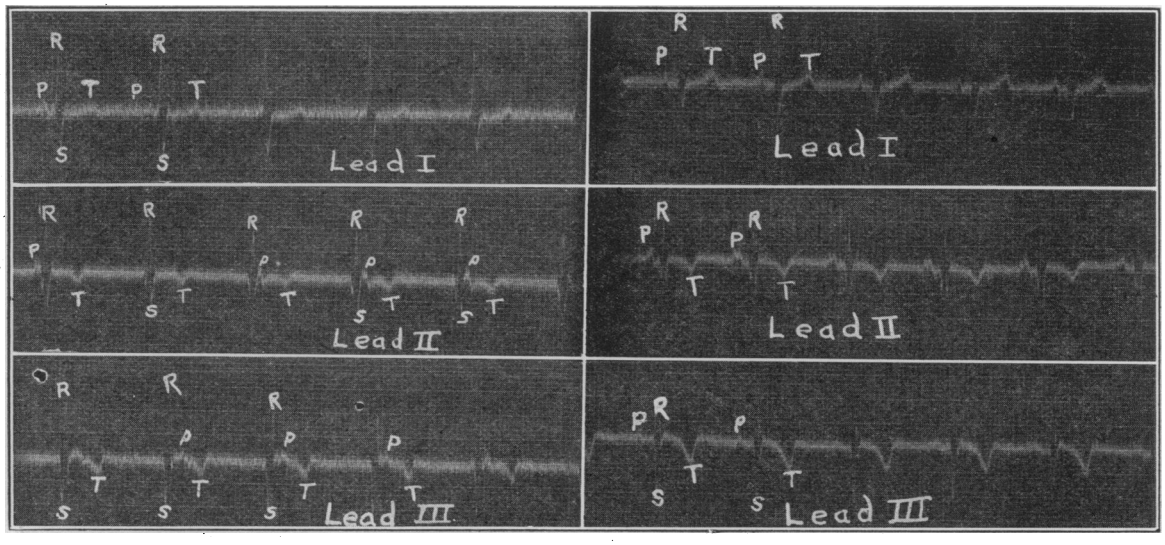

Fig. 1a (Case 1)

Fig. 1b (Case 1)

tracing was returned. The patient was a man who had always been in good health except for occasional attacks of what he referred to as indigestion. There had been no attacks of precordial pain but frequent attacks of pain on the right side below the diaphragm, and some other evidences of gallbladder inflammation. In April 1926, there was an attack slightly more severe than at any previous time and again in July 1926, during which time there was some precordial pain. In August an attack became so severe that it left no doubt that the pain was cardiac in origin. He was taken to the hospital and an electrocardiogram showed a decided inversion of the T wave in all three leads and a high grade incomplete A-V block (Fig. 2b). Two months of absolute rest in bed was carried out, with the patient in the orthopneic position. Caffein was given routinely and morphin grains one-quarter on two occasions. During two months there was no recurrence of pain in the chest and the patient felt quite well except for occasional attacks of palpitation. On examination the blood pressure was 130/90, pulse 78, temperature 101, and the white blood count 14,500. Another electrocardiogram taken at the end of two months showed a restoration of the normal P-R interval, inversion of the P waves, but the T wave had become positive again in all leads (Fig. 2c). Frequent observation of the patient during the last two months reveals occasional slight attacks of abdominal discomfort, and a history of mild atonic colitis with belching of gas.

Case 3.—Female, age sixty-six. On the eve of April 2, 1927, the patient was seized with a sharp stabbing pain in the chest which continued during most of the night. She was seen at 7 a. m. the following morning, and during examination suddenly felt that she was dying. She lost consciousness, became pale as in death, had a convulsion, became pulseless and apparently was dead.

A hypodermic injection of 20 minims of adrenalin was given intravenously, but probably before it had time to act the patient became conscious again and the pulse became perceptible. The heart action was increased in force and the rate quickly rose to 100 per minute. The patient was removed to a hospital, where the electrocardiogram showed a high grade incomplete heart block with inversion of the T wave in all three leads. The coronary arching was particularly well shown in the second and third leads, associated with left ventricular preponderance and severe myocardial damage (Fig. 3a). On physical examination the blood pressure was 100/90, temperature 99 to 100 during the first week, pulse 40, and white blood count 16,400.

This patient was kept at absolute rest in the orthopneic position for two weeks and the usual treat-

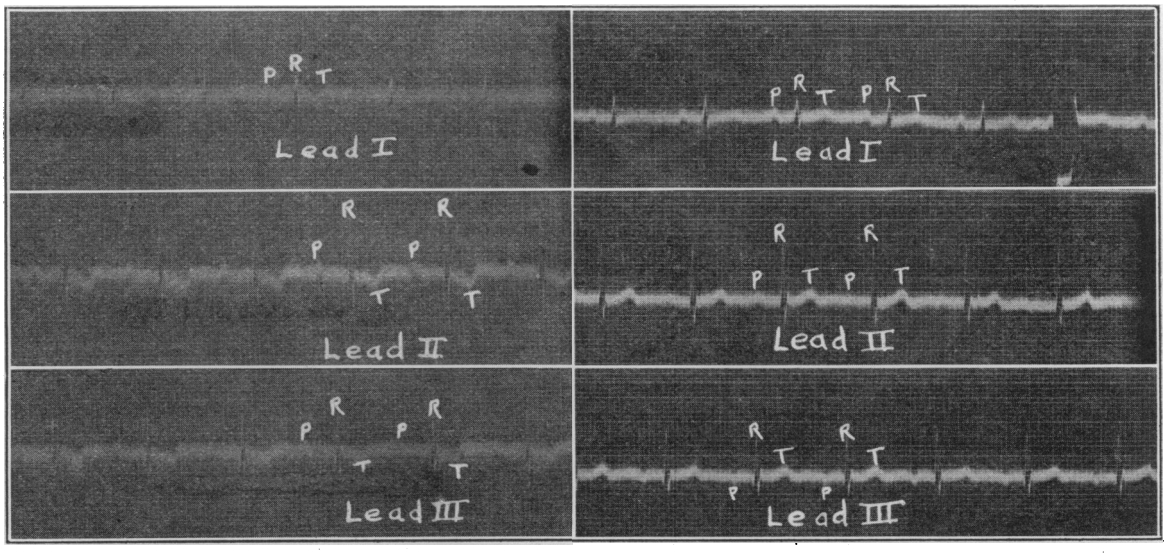

Fig. 2b (Case 2)

Fig. 2c (Case 2)

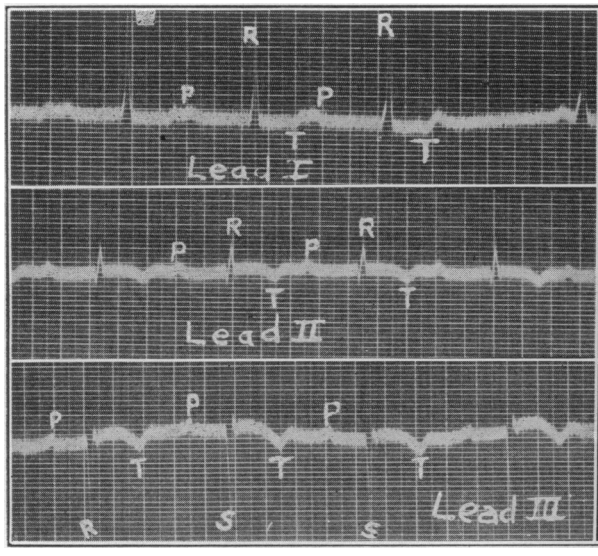

Fig. 3a (Case 3)

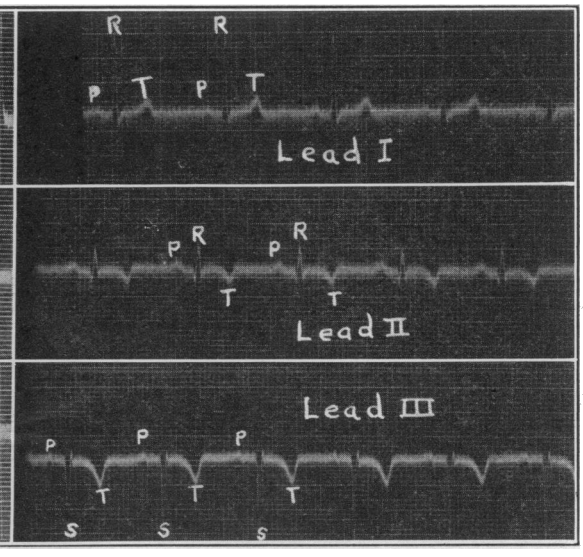

Fig. 3b (Case 3)

ment as given above. During this time there was never any recurrence of precordial pain or cyanosis, but occasional attacks of palpitation. After six weeks of further rest in bed at home, another electrocardiogram was taken which revealed restoration of the normal sinus rhythm with disappearance of the A-V block. The T wave is definitely positive in Lead I, but inversion still remains in Leads II and III (Fig. 3b).

* * *

Case 4.—Male, age forty-nine. This patient was first observed in March 1927, while in the hospital for an acute toxemia. There had been some precordial distress for several weeks, but no definite pain or dyspnea. Palpitation had been a feature. In October 1927, the patient was seen again, after a severe attack of pain in the chest which radiated to both arms and under the left shoulder. This attack came on immediately after climbing a flight of stairs. On examination he was quite pale, perspiring freely, slightly cyanosed and perceptibly short of breath. The pulse was soft and compressible, irregular and 100 per minute. The heart tones were indistinct and no murmurs were heard either at the apex or base. The rhythm was interrupted every fourth beat by premature contractions and the blood pressure was 132/98. The heart was not enlarged to percussion. After four weeks of rest in bed, the patient was transferred to the hospital where an electrocardiogram revealed inversion of the T wave in the second and third leads with a suspicion of the coronary curve associated with notching of the Q R S complex in all leads and left axis deviation (Fig. 4). This man is now attending to his practice, but limiting his work to office consultation, and has had no severe attacks during the last three months.

* * *

Case 5.—Male, age sixty-three. The patient was seen on May 2, 1927, several hours after a severe attack of precordial pain. The attack lasted about an hour until a physician was called who recognized the symptoms as cardiac, and administered one-quarter grain of morphin sulphate. Relief was obtained almost immediately. The patient had been healthy until the last few months, when several attacks of precordial distress were noticed, particularly in the early morning.

On examination the lips were slightly cyanosed, the skin pale, pulse 80, blood pressure 132/80, vital capacity 62 per cent. There was slight enlargement of the heart to the left, no apical or basal murmurs and no cardiac irregularity.

Rest in bed for four weeks was advised and during this time there was no recurrence of symptoms. Following the rest period an electrocardiogram revealed inversion of the T wave in the second and third leads and notching of the Q R S complex in all leads (Fig. 5). The patient has been quite comfortable for eight months, but has restricted his activity considerably and takes small doses of theobromin daily.

CLINICAL COURSE

The clinical picture presented by the above cases is that of severe collapse associated with intolerable pain in the chest, referred to one or both arms, boring, knife-like, tearing or gripping, and sometimes referred to as a choking pressure. Nausea and vomiting may be present. Cyanosis is usual and may be associated with pallor and cold sweats. Fever and leukocytosis are frequently found during the first few days. Dyspnea is a prominent feature and eventually edema of the lungs may develop with spitting of blood. Convulsions may occur (Case 3). Physical examination is frequently of little aid. The heart may or may not be enlarged, and the blood pressure and pulse are variable factors. Occasionally alternation of the pulse is found and the heart tones are frequently very poorly heard and there may be no murmurs. A pericardial friction rub is of diagnostic importance, but is not always heard. When present it is more apt to be heard twenty-four hours after the onset.

In most instances a large coronary artery is occluded and death is instantaneous or at least supervenes within a few hours. In the cases mentioned above, the onset and symptoms were most severe, but instead of collapse and death the patients gradually improved and all are now living and well, after periods varying from two months to a year. Probably a small arteriole was occluded in these instances and small areas of infarction formed which were not large enough to embarrass the heart sufficiently to cause death.

PATHOLOGY

The pathology is generally that of sclerosis and atheroma of the coronary vessels. The descending branch of the left coronary is more seriously involved in the majority of cases. Infarcts gener-

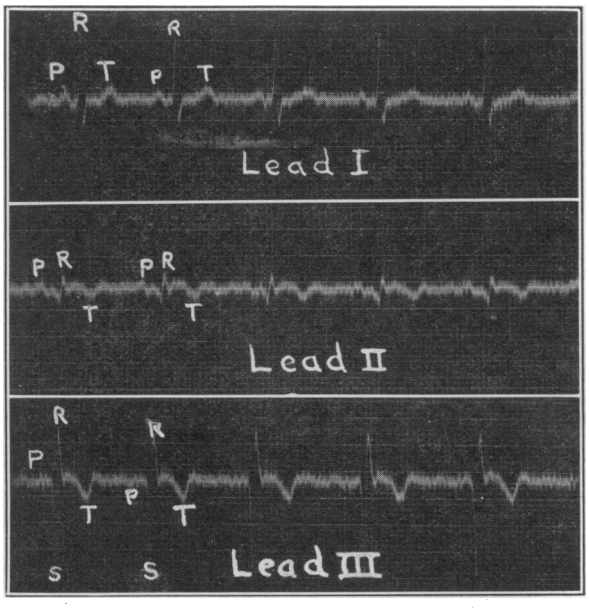

Fig. 4 (Case 4)

ally occur in the anterior two-thirds of the left ventricle. If the area of infarction has been large and the inflammation extensive, the pericardium may be found adherent at the site of infarction.

Fatty degeneration, so-called, is not a factor in the production of these changes. Probably the fatty degeneration, so frequently referred to in the past, is merely the myocardial degeneration resulting from coronary sclerosis with small occlusions. At autopsy the heart will generally show some hypertrophy.

Certain etiological factors should be considered in passing. The causative factors of arteriosclerosis and hypertension are frequently those of this condition. The most important chronic infections include syphilis, rheumatic carditis, and ulcerative endocarditis. Minor factors which may be responsible for a precipitation of the symptoms are: extreme physical exertion calling for a sudden powerful contraction of the heart, such as the carrying of heavy weights, coughing, defecation, coitus. Great excitement and emotional upsets, and overeating should also be placed in this category.

COMMENT

The cases given above confirm the diagnosis of coronary artery disease both clinically and from the standpoint of the electrocardiograph. In only one case had digitalis been given, that of Case 2, and in this case no digitalis had been taken within one week of the electrocardiographic tracing. A definite inversion of the T wave is found in the records of certain individuals who have been taking this drug, and the effect is still frequently present even after ten days. The fact that a later tracing showed a normal picture may indicate first, that the attack was an acute coronary spasm, and second, that digitalis was actually a disturbing factor in the electrocardiographic tracing.

It would seem from a study of these cases that coronary artery disease gives rise to a certain clinical picture and should be more easily recognized in the future by the aid of the electrocardiograph.

When an individual past forty years of age has an attack of so-called acute indigestion following a heavy meal or unusual physical or mental exercise, the possibility of coronary artery disease must first be considered. The diagnosis can be made only after a careful history and complete cardiac study.

1052 West Sixth Street.

DISCUSSION

HARRY SPIRO, M. D. (870 Market Street, San Francisco).—I find it more than ordinarily difficult to add any information to what Doctor Langley has already stated because in a more than ordinarily concise manner he has covered the subject of coronary artery disease. Furthermore, I would like to congratulate him on his results. He is far more fortunate than I am.

The questions of diagnosis as Doctor Langley has explained them will always be of prime importance. It surely must be disconcerting to the conscientious physician to make a diagnosis of acute indigestion and then have his patient suddenly die. The differential diagnosis on the other hand, between coronary artery thrombosis and acute gall-bladder disease, is not always easy and a mistake is forgivable, especially so if one makes a diagnosis of coronary artery thrombosis when it is only an acute gall-bladder disease. The most disconcerting thing about the whole question of diagnosis of coronary artery thrombosis is the indefiniteness of a physical examination, as Doctor Langley has pointed out. The heart may be of average size, the rhythm of the heart, particularly as found at the radial artery, may be perfect, the heart sounds may be clear, and yet the patient be in imminent danger of death. This is what makes the history of such marked importance. Here is the one case in which, if the symptoms are typical, all laboratory tests, physical examination, x-ray examinations, or electrocardiograms, may be disregarded to let the patient's statements rule. The proof of the above statement is the fact that a patient may have the symptoms typical of coronary artery stroke such as described by Doctor Langley, may die a cardiac death, and yet postmortem examination disclose very little. One of the principal reasons is that these patients may die because a very small branch of the coronary artery may be suddenly occluded and this sudden occlusion so irritate some part of the ventricular muscle as to produce ventricular fibrillation and thus instant death. I believe it is generally recognized that the most frequent cause of death in angina pectoris is ventricular fibrillation.

The question as to whether one should use digitalis or vasodilators is another problem which is under active discussion. I believe that if a physician is actually convinced that a patient has an acute coronary artery stroke, particularly if there is associated

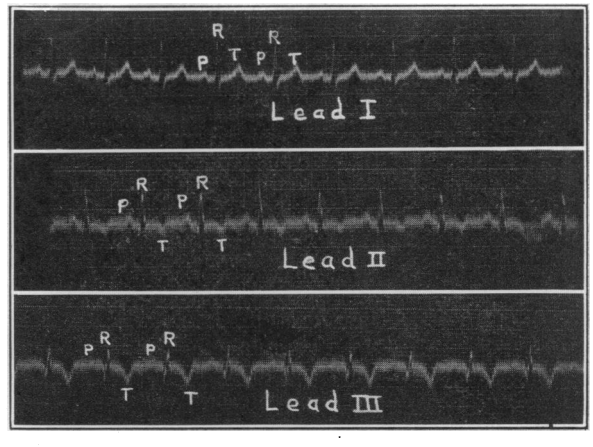

Fig. 5 (Case 5)

with the symptoms a marked fall in blood pressure, that he is then justified in the use of digitalis. If the heart can be supported and the circulation maintained long enough, there is a chance for the patient to make a recovery. On the other hand I feel that if the patient gets any relief from the use of vasodilators, these should be used either alone or in conjunction with digitalis. I regard it as particularly ominous for a patient who has had symptoms of coronary artery stroke to develop an arrhythmia of any type during the acute phase. These patients appear to be doing fairly well, have no more pain, and die very, very suddenly.

Another dangerous type is one in which the patient has recovered from his attack of pain, feels more than ordinarily well, has no distress of any kind, and yet his pulse rate is fast, over 100. These patients frequently drop dead the first time you let them stand up or go to the toilet. If a patient has had symptoms of coronary artery stroke, has apparently recovered and in the first few weeks of his recovery has a fast pulse rate, over 110, keep him in bed.

Shall a man smoke or shall he not? I believe nicotin is a cardiac muscle irritant; for that reason it is probably best for the patient to discontinue the use of tobacco and only resume its use very gradually, if at all.

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FRANKLIN R. NUZUM, M. D. (Santa Barbara Cottage Hospital, Santa Barbara.—I, too, would like to add my commendation of Doctor Langley's paper. I would like to add a further word concerning the changes in the heart muscle that follow an acute blocking of one of the coronary vessels. An anemic infarct is the result. The size of the infarcted area depends on the size of the occluded vessel. A very firm, strong heart muscle within twenty-four to forty-eight hours following an occlusion becomes at first light yellow in color and later dark red; its softness is surprising, and the ease with which one may thrust his finger or a blunt probe through such an infarcted area makes one wonder why many more of these patients do not die of a rupture through the infarcted area.

In some more than six hundred reported instances in the literature, of rupture of the heart wall, the area of infarction has, in over 95 per cent, involved the anterior descending branch of the left coronary artery, *i. e.*, that vessel which supplies the anterior wall of the left ventricle and a portion of the ventricular septum. In these six hundred instances death resulted from a rupture through the infarcted area, and the exact location of the infarct and the obstruction in the coronary artery were demonstrated by postmortem examination. Since the anterior wall of the left ventricle is usually infarcted, the fibrinous adhesions which form over this area rub upon the anterior surface of the pericardial sac, producing a friction sound. This friction, when searched for, is very frequently present. It develops ten to eighteen hours after the occlusion of the coronary vessel. It usually disappears within ten to twenty-four hours after it first becomes audible. When present it is the most striking point in the physical examination in proving the diagnosis of a coronary occlusion. Temperature, a leukocytosis, and a so-called coronary T wave in the first or second lead of the electrocardiogram complete the clinical findings and indicate a large area of infarction of the myocardium.

When small branches of the coronary arteries are occluded, the areas of infarction may be minute. These instances are often more difficult of diagnosis, but are equally important clinically, since, as has been stated, ventricular fibrillation may follow, and this type of disturbed rhythm results in death.

That rupture of large infarcted areas occurs more commonly than was generally believed is becoming recognized. I personally have nine pathological specimens in which such a rupture occurred. In each of these instances the patient had gotten out of bed within twenty-four to forty-eight hours following the vessel occlusion and death was sudden.

The ability of nature to repair such an infarcted area is equally impressive. Large, firm, fibrous scars, involving particularly the left ventricular wall, in some instances measuring several centimeters in length and breadth, attest to the importance of keeping such a patient at bed rest until complete fibrosis has had time to take place.

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EUGENE S. KILGORE, M. D. (490 Post Street, San Francisco).—Clinical interest in coronary thrombosis is rightly focused mainly on diagnosis; and it is gratifying that intelligent study now usually establishes the diagnosis during life, whereas a few years ago it was very exceptional for this to occur. Doctor Langley has illustrated the value of electrocardiography and the usual symptoms and physical signs.

With little more additional data the electrocardiogram or the pericardial friction sound may establish the diagnosis. More frequently, however, a careful study of symptoms alone will be nearly or quite decisive, and conversely, and which is most important, a superficial attention to symptoms is usually responsible for the occasional costly error of mistaking coronary thrombosis for an acute abdominal condition.

Pain is often absent, or it may be mild or excruciating. Most commonly substernal, it may be precordial, diffusely over chest and back, or occasionally only epigastric. It may or may not radiate—usually to the left arm, especially under the arm to the elbow, wrist or radial distribution in the hand; or to the right arm or both arms, the neck, jaw, or occiput. It is variously described (according to preconceptions of the patient) as "pleuritic," "indigestion," etc.; but more particular questioning will often bring out the quality of pressure—"vise-like," "constriction about the windpipe," "internal gas pressure," "petrified feeling," etc., or it may be simply indescribable. It is not colicky. It is not lightning-like in onset, but usually has a distinct crescendo period. It may be worse after eating and somewhat relieved (if not too severe) by belching. It is likely to come on without effort and be unrelieved by rest, by nitrites, and by small or moderate doses of morphin. Especially in the cases with pain under the lower sternum or in the epigastrium, belching is frequent, and nausea and vomiting not uncommon. These symptoms, with pain, fever, and leukocytosis, create at times a very perplexing diagnostic problem; and it is here especially that a careful scrutiny of the heart by all methods including the electrocardiograph is most important.

THE SURGERY OF TUMORS OF THE BLADDER*

By FRANK HINMAN, M. D.
San Francisco

DISCUSSION *by Benjamin H. Hager, M. D., Los Angeles; James F. Percy, M. D., Los Angeles; R. L. Rigdon, M. D., San Francisco.*

BEFORE 1910, when Edwin Beer introduced fulguration, the only treatment available for a bladder tumor was surgical, and the results were poor and discouraging for both malignant and benign growths. There have been no epochal advances of surgical technique since then. Nevertheless we are able today to accept the responsibility of treatment of such a case with a certain sense of confidence which, however, is far from complete. The newest agent, radiation, is bolstered high above its ability by a hopeful enthusiasm, and we seem now to have reached a dead level of advance in the cure of vesical malignancy.

TREATMENT METHODS

Perhaps the next rise will follow improvement of surgical method, but discussion of this is pos-

* Read before the Urological Section of the San Francisco County Medical Society, January 29, 1929.